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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/673,038	09/29/2003	Verena Grimm	035642-0104	8031
22428 7590 02/26/2007 FOLEY AND LARDNER LLP SUITE 500 3000 K STREET NW WASHINGTON, DC 20007			EXAMINER POHNERT, STEVEN C	
			ART,UNIT	PAPER NUMBER
			1634	

SHORTENED STATUTORY PERIOD OF RESPONSE	MAIL DATE	DELIVERY MODE
3 MONTHS	02/26/2007	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

If NO period for reply is specified above, the maximum statutory period will apply and will expire 6 MONTHS from the mailing date of this communication.

Office Action Summary	Application No. 10/673,038	Applicant(s) GRIMM ET AL.	
	Examiner Steven C. Pohnert	Art Unit 1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 20 December 2006.
- 2a) ☒ This action is **FINAL**. 2b) ☐ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 1-14 is/are pending in the application.
- 4a) Of the above claim(s) 14 is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 1-13 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☒ The drawing(s) filed on 9/29/2003 is/are: a) ☒ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) ☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☒ All b) ☐ Some * c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
 2. ☐ Certified copies of the priority documents have been received in Application No. _____.
 3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- | | |
|--|---|
| 1) <input type="checkbox"/> Notice of References Cited (PTO-892) | 4) <input type="checkbox"/> Interview Summary (PTO-413)
Paper No(s)/Mail Date. _____ |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948) | 5) <input type="checkbox"/> Notice of Informal Patent Application |
| 3) <input type="checkbox"/> Information Disclosure Statement(s) (PTO/SB/08)
Paper No(s)/Mail Date _____ | 6) <input type="checkbox"/> Other: _____ |

DETAILED ACTION

This action is in response to the papers filed on 12/8/2006. Currently claims 1-14 are pending. All arguments have been thoroughly reviewed but are not deemed to be persuasive for the reasons that follow.

This action is FINAL.

Any objections and rejections not reiterated below are hereby withdrawn.

Maintained rejections

1. Applicant's election with traverse of group I, claims 1-13 in the reply filed on June 5, 2006 is acknowledged. The traversal is on the ground(s) that the plurality of sets of nucleotide sequences of the general formula R1- (X)-R2 are structurally related. This is not found persuasive because, the general formula R1- (X)-R2 can apply to any nucleotide sequence in any gene or genome, and is not specific to the beta lactamase gene. The composition and sequence of individual nucleotides comprising a nucleotide sequence determines the structure of the nucleotide sequence. The general formula R1- (X)-R2 does not specify a specific nucleotide sequence or composition, but rather a genus of sequences, the species of this genus has specific nucleotide composition and are thus structurally distinct. The structure and composition of probes listed in Table 2 of the specification demonstrate distinct chemical compositions and sequences, and as such are patentably distinct.

Election/Restrictions

Applicant's states," contrary to the examiner's assertion, the formula R1-(X)-R2 does not ' apply to any nucleotide sequence in any gene or genome." Rather, R1-(X)-R2 are derived from the beta-lactamase gene."

Applicants arguments filed 12/8/2006 are fully considered, but are not found persuasive. Although each probe depicted by R1-(X)-R2, is derived from the beta-lactamase gene each probe encompassed by the R1-(X)-R2 formula has a distinct nucleotide sequence and chemical composition. Each probe thus detects a distinct nucleotide sequence and thus is not obvious over the other probes that are depicted by the formula.

The office action mailed on 8/9/2006, made this restriction final. As the restriction has been made final, the applicant can further contest the validity of the restriction by petition. However, if the generic claim is found to be allowable the species can be rejoined at that time.

Claim 14 is withdrawn from further consideration pursuant to 37 CFR 1.142(b), as being drawn to a nonelected invention, there being no allowable generic or linking claim. Applicant timely traversed the restriction (election) requirement in the reply filed on 12/8/2006.

This application contains claim 14 drawn to an invention nonelected with traverse in Paper No. 12/8/2006. A complete reply to the final rejection must include cancellation of nonelected claims or other appropriate action (37 CFR 1.144) See MPEP § 821.01.

Claim Rejections - 35 USC § 103

2. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

3. Claims 1-6, 8, 9, 11, and 13 are rejected under 35 U.S.C. 103(a) as being unpatentable over Lee, et al (Molecules and Cells (2002) volume 14, pages 192-197) in view of Blazquez, et al (Antimicrobial Agents and Chemotherapy (1995) volume 39, pages 145-149), Chee et al (A) (WO 95/11995), and Sutcliffe (Proceedings National Academy of Sciences USA (1978), volume 75 pages 3737-3741).

Claim 1 is drawn to obtaining a biological sample, optionally isolating and/or amplifying DNA from the sample and contacting the DNA from the sample with an array with capture probes derived from the sequence of a beta-lactamase genes to determine

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the presence of a beta lactamase resistant organism and mutations indicative of resistance to specific antibiotics.

With regards to claims 1, Lee teaches the use of arrays in which hybridization is indicative of beta-lactamase resistance (see abstract). Lee further teaches hybridization of target DNA with a DNA-chip, or micro-array, with probe sets at specific locations on the chip (see page 93 hybridization, preparation of DNA chip) to determine the presence of beta lactamase resistant genes (see figures 1, 3, 4) using probes of 21 nucleotides.

With regards to claims 2 and 3, Lee teaches isolation and amplification of target DNA from bacterial cells prior to contacting with array (see page 193, last paragraph 1st column and 1st paragraph 2nd column).

With regards to claims 8, Lee et al teaches a micro-array for the detection of various beta-lactamase resistant genes, including PSE, OXA, FOX, MEN, CMY, TEM, SHV, OXY, and *AmpC* (see abstract).

With regards to claim 11 and 13, Lee teaches the fluorescent labeling of DNA prior to contact with array (see page 193, column 1 last paragraph 3rd line up). Lee et al does not teach a set of probes with all combinations of probes comprising R1- (X)-R2 of a beta lactamase gene (claim 1), fragmentation of DNA prior to contacting it with array (claim 4), beta-lactamases from Enterobacteriaceae (claim 5), or known SNPs in the beta-lactamase gene (claim 6).

However, with regards to claims 1,6, Blazquez et al teaches mutations, or SNPs, of beta-lactamase, including the mutation of gln39lys (specification refers to gln37lys) (see figure 1) alter beta lactamase stability and antibiotic activity (see page 148 column

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1, 1st 4 lines of next to last paragraph). (It is noted that there are two accepted number systems for beta-lactamases, the Ambler numbering system based used by Blazquez and the Sutcliffe system used in the specification. The two numbering systems may stem from Sutcliffe's sequencing pBR322 and Ambler sequencing the protein product of R6K (Proceedings National Academy of Sciences, USA (1978), Volume 75 pages 3732-3736). The art are normally presents mutants in reference to either Ambler or Sutcliffe numbering. The specification does not explicitly state a numbering system. Neither numbering system has glu at both codon 37 and 39. It is thus presumed codon 37 of specification and codon 39 of Blazquez are the same barring proof otherwise.)

Blazquez further teaches introduction of known mutations into the TEM1 gene at codons 39, 104, 164, 237, 238, and 240 alter resistance of microorganisms to specific antibiotics (see table 2 and 3). Mutation of amino acid 39 specifically decreases the susceptibility to cephaloridine and ceftazidime, but not amoxicillin, amoxicillin plus clavulanic acid, cefotaxime, aztreonam, and meropenem (see tables 2 and 3, and page 146 2nd column paragraph (i).

With regards to claim 5, Blazquez teaches E.coli which is a member of the Enterobacteriaceae family.

With regards to claims 1 and 4, Chee (A) et al teaches a tiling array (see Figure 7 and page 37 line 10- page 38 line 34). Chee (A) teaches the use of immobilized arrays to interrogate a reference sequence and its codons with a target sequence for the identification of single base mutants in the reference sequence associated with disease (see page 31 lines 6-7, and page 11 line 9 and 10). Further Chee (A) teaches this

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approach allows simultaneous detection and quantification of multiple target sequences (see page 32 lines 18-19), allowing for sequence determination. The block-tiling array allows the interrogation of multiple nucleotide sites by use of multiple probe sets, which represent every permutation of nucleotides possible for a given nucleotide sequence. Chee (A) teaches the determination of all possible combinations of nucleotides surrounding a SNP using from 15-30 nucleotides (page 27 lines 2-6), allowing determination of all possible nucleic acid sequences. With regards to claim 4, Chee et al teaches DNA fragmentation (see page 126, number 4), prior to contacting with capture probes. Chee (A) teaches microfabricated arrays with large numbers of oligonucleotides offer great promise (see page 2 lines 11-13) for applications including identification of mutations related to disease, forensic studies, epidemiological and forensic studies. Chee (A) further teaches, "It is desirable to simultaneously diagnose the presence or absence of a variety of lethal common infections, determine the most effective therapeutic regime" (see page 64 lines 25-28).

With regards to claims 1 and 6, Sutcliffe teaches the nucleotide sequence of the beta-lactamase gene (see figure 3).

Therefore, it would be prima facie obvious to one of ordinary skill in the art at the time the invention was made to improve the method of identifying beta-lactamase resistance taught by Lee, to include identification of specific mutations resulting in resistance to specific antibiotics as taught by Blazquez. The ordinary artisan would be motivated to combine the teaching of Lee and Blazquez in order to identify the specific beta lactam resistance present and provide proper treatment. It would have been

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further prima facie obvious to the ordinary artisan at the time the invention was made to improve the method of identifying specific mutations for specific resistance of Lee and Blazquez to incorporate a tiling array as taught by Chee (A) and use the beta lactamase sequence taught by Sutcliffe to allow simultaneous detection and quantification of multiple target sequences (see page 32 lines 18-19), thus allowing thorough characterization of mutations known to alter beta lactam resistance, and possibly identify new mutations altering beta-lactam resistance. The ordinary artisan would be motivated to improve the method of Lee and Blazquez with the use of a tiling array as taught by Chee (A), because Chee (A) teaches simultaneously diagnose the presence or absence of a variety of lethal common infections, determine the most effective therapeutic regime (see page 64 lines 25-28). In performing the method of Lee, Blazquez, Chee (A), and Sutcliffe, the ordinary artisan would be motivated to specifically include a probe in the screening method, corresponding to the mutation at codon 37 (same as codon 39 taught by Blazquez), because Blazquez teaches that a mutation at this codon results in specific antibiotic resistance. The ordinary artisan would be motivated combine the teachings of Lee, Blazquez, Chee (A), and Sutcliffe to screen for the presence of mutations at codon 37 (codon 39 taught by Blazquez) with the use of tiling probes as taught by Chee (A) to detect the susceptibility to cephaloridine and ceftazidime. The ordinary artisan would therefore be motivated to construct a probe corresponding to mutations in codon 37, including a probe with the sequence of SEQ ID NO: 7 (claim 9), to provide tiling probes with all possible combinations of nucleotides at codon 37.

Response to Arguments

4. The response filed 12/8/2006, asserts on page 8 that Blazquez, Sutcliffe, and Chee do not teach the permutations of the selected sequences of the beta-lactamase gene or probes that start 3n positions downstream of the first probe set. The response on page 9, first full paragraph further asserts that examiner failed to provide motivation for the combination of the micro-array of Lee with the tiling array of Chee (A).

Applicant's arguments have been fully considered but they are not persuasive.

With respect to the arguments directed to probes starting 3n positions downstream, the MPEP 2111.03 states, "The transitional term "comprising", which is synonymous with "including," "containing," or "characterized by," is inclusive or open-ended and does not exclude additional, unrecited elements or method steps." Chee teaches an incremental succession of probes in a basic tiling strategy in which each probe differs from its predecessor by the acquisition of a 5' nucleotide and the loss of a 3' nucleotide as well as the nucleotide occupying the interrogation position (see page 11, lines 21-27 and figure 3). Chee teaches this tiling method allows for the detection of mutations or polymorphisms (see page 18, lines 8-10). Chee further teaches the use of probes are from 9 to 30 nucleotides, which is in the range of 9 to 43 nucleotides depicted by the formula R1-(X)-R2, in which R1 and R2 are each 3 to 20 nucleotides (see page 27, lines 4-5). Therefore Chee teaches a basic tiling array for detection of polymorphisms, which comprises the probe sets that start at position 3n of the nucleotide downstream of the first probe set.

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The nucleotide sequence of the beta-lactamase gene taught by Sutcliffe (see Figure 3) would serve as the reference sequence for the incremental succession of probes taught by Chee, while mutations taught by Blazquez would direct the ordinary artisan to specifically examine triplets specifically known to result in beta lactam resistance. Further, the sequence taught by Sutcliffe is one permutation of the nucleic acid sequence, while the sequence in table 1 of Blazquez is another sequence resulting in beta lactamase resistance. The ordinary artisan would thus have the nucleotide sequence with the aligned amino acid sequence taught by Sutcliffe to readily determine the nucleic acids involved in each taught by Blazquez to result in beta lactam resistance use in Chee's method of tiling.

With regards to the assertion that the examiner did not provide motivation for combination of Lee with the tiling array of Chee, the examiner notes from page 8 of reply file 12/8/2006, "to allow simultaneous detection and quantification of multiple target sequences (see page 32 lines 18-19), thus allowing thorough characterization of mutations known to alter beta lactam resistance, and possibly identify new mutations altering beta-lactam resistance." Thus designing a tiling array to analyze the known beta lactamase gene would have been obvious.

5. Claim 7 is rejected under 35 U.S.C. 103(a) as being unpatentable over Lee, Blazquez, Chee (A) and Sutcliffe, as applied to claims 1-6, 8, 9, 11, and 13 above, in further view of Osano et al (Antimicrobial Agents and Chemotherapy (1994) volume 38, pages 71-78)

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Claim 7 further limits the enterobacteriaceae beta lactamase of claim 5 to a serine or zinc beta-lactamase.

The teachings of Lee, Blazquez, Chee (A) and Sutcliffe are set forth above. Lee, Blazquez, Chee (A), and Sutcliffe, do not teach on serine or zinc beta-lactamase from the bacterial family Enterobacteriaceae.

However, Osana et al, teaches *S. marcescens* is a member of enterobacteriaceae family (see page 76 column 1, lines 16-19). Osana further teaches class A and C beta-lactamases, including plasmid encoded TEM and SHV, are serine dependent (see page 71, column 1, lines 5 and 6 column 1 and column 2 lines 4-7). Osana further teaches class B beta-lactamases are zinc dependent (see page 71 column 2 lines 4-7). Osana teaches that some strains of the Enterobacteriaceae family are reported to be resistant to imipenem therapy. Osana teaches IMP-1 a zinc beta lactamase confers imipenem resistance to *S. marcescens* and further demonstrates there is great variability in the amino acid sequence of known zinc beta-lactamases and suggests evolution independent of other known zinc beta lactamases (see figure 3).

Therefore it would have been prima facie obvious to the ordinary artisan at the time of the invention was made, to improve the method taught by Lee, Blazquez, Chee (A) and Sutcliffe, to include the zinc beta-lactamases as taught by Osano, et al for the purpose of detecting imipenem resistance in the Enterobacteriaceae family. The ordinary artisan would be motivated to use the method taught by Lee, Blazquez, Chee (A), and Sutcliffe to determine the presence of zinc beta lactamases in order to identify imipenem resistant strains, to allow for proper diagnosis and treatment.

Response to arguments

6. Applicant asserts in the reply filed 12/8/2006 for reasons discussed above that Chee, Sutcliffe, Lee and Blazquez do not meet the limitations of the independent claims and thus do not cure the defects of claim 7.

Applicant's arguments have been fully considered by they are not persuasive. The response asserts Claim 7 depends from Claim 5 and ultimately Claim 1. The response states that Lee, Blazquez and Chee (A) are discussed above and Osono does not cure the defects of the primary or secondary references. This argument is not persuasive for the reasons presented above for Lee in view of Blazquez and Chee (A) and Sutcliffe.

7. Claim 10 is rejected under 35 U.S.C. 103(a) as being unpatentable over Lee, Blazquez, Chee (A), and Sutcliffe as applied to claims 1-6, 8, 9, 11, and 13, further in view of Chee (B) et al (Science (1996), Volume 274, pages 610-614) and Routier (Nucleic Acids Research, (1999) volume 27, pages 4160-4166).

The teachings of Lee, Blazquez, Chee (A) and Sutcliffe are set forth above. Lee, Blazquez, Chee (A), and Sutcliffe do not teach fragmentation of DNA to 15-50 nucleotides.

However, Chee (B) teaches fragmentation improves the uniformity and specificity of hybridization (see page 613 third column, lines 43 and 44). Routier teaches a method of fragmentation resulting in fragments of 15-50 nucleotides (see Figure 5).

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Therefore it would be prima facie obvious for one of ordinary skill the art at the time of the invention to modify the method of Lee, Blazquez, Chee (A), and Sutcliffe for detection of beta lactamase resistance with the Routier method of DNA fragmentation wherein the fragments are 15-50 nucleotides. Routier teaches fragmentation with sizes of 15-50 nucleotides and Chee (B) teaches fragmentation improves uniformity and specificity of hybridization. The ordinary artisan would be motivated to optimize the size of fragments of the DNA prior to contacting with a microarray because Chee (B) teaches it improves specificity and uniformity of hybridization.

As stated in the MPEP, 2144.05 II, "[W]here the general conditions of a claim are disclosed in the prior art, it is not inventive to discover the optimum or workable ranges by routine experimentation." In re Aller, 220 F.2d 454, 456, 105 USPQ 233, 235 (CCPA 1955)."

Response to arguments

8. Applicant asserts in the reply filed 12/8/2006 for reasons discussed above that Chee, Sutcliffe, Lee and Blazquez do not meet the limitations of the independent claims and thus do not cure the defects of claim 10.

Applicant's arguments have been fully considered by they are not persuasive. The response asserts Claim 10 depends from Claim 4 and ultimately Claim 1. The response states that Lee, Blazquez and Chee (A) are discussed above and Chee (B) and Routier do not cure the defects of the primary or secondary references. This argument is not persuasive for the reasons presented above for Lee in view of Blazquez and Chee (A) and Sutcliffe.

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9. Claim 12 is rejected under 35 U.S.C. 103(a) as being unpatentable over Lee, Blazquez, Chee (A), and Sutcliffe as applied to claims 1-6, 8, 9, 11, and 13 above, and further in view of Behrendorf, et al (Nucleic Acids Research (2002) volume 30, e64).

The teachings of Lee, Blazquez, Chee (A) and Sutcliffe are set forth above. Lee, Blazquez, Chee (A) and Sutcliffe do not teach labeling of DNA following contacting DNA with array.

However, Behrendorf, et al teaches the detection of SNPs by binding fluorescently labeled mutS to mismatched DNA for detection of SNPs (see figure 1) on an array. Behrendorf teaches this method allows, "robust detection of genetic variation," while decreasing hybridization times and shortening assay duration (see page 5, 1st column lines 1 and 2, and 2nd column lines 10-12).

Therefore it would be prima facie obvious to the ordinary artisan at the time the invention was made to improve the array based method of detecting the presence of beta-lactam resistant bacteria of Lee, Blazquez, Chee (A) and Sutcliffe by using fluorescently labeled mutS taught by Behrendorf to label the DNA after contacting with array, because Behrendorf teaches that it gives robust detection of genetic variations, decreases hybridization times and shorten assay duration. The ordinary artisan would be motivated to use the labeling method of Behrendorf in the method of Lee, Blazquez, Chee (A) and Sutcliffe because Behrendorf teaches that it gives robust detection of genetic variations, decreases hybridization times and shorten assay duration.

Response to arguments

10. Applicant asserts in the reply filed 12/8/2006 for reasons discussed above that Chee, Sutcliffe, Lee and Blazquez do not meet the limitations of the independent claims and thus do not cure the defects of claim 12.

Applicant's arguments have been fully considered by they are not persuasive.

The response asserts Claim 12 depends from Claim 1. The response states that Lee, Blazquez and Chee (A) are discussed above and Behrendorf does not cure the defects of the primary or secondary references. This argument is not persuasive for the reasons presented above for Lee in view of Blazquez and Chee (A) and Sutcliffe.

Summary

No claims are allowed over prior art cited.

Conclusions

11. Applicant's arguments have been fully considered but they are not persuasive. Thus for the reasons above and those already of record, the rejection is maintained

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Steven C. Pohnert whose telephone number is 571-272-3803. The examiner can normally be reached on Monday-Friday 7:00-3:30.

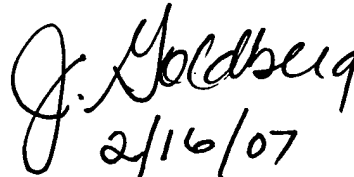
If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla can be reached on 571-272-0735. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

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Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.



Steven Pohnert


2/16/07
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